PAIN

From Neuroscience to Neurofeedback and Neuromodulation

Insights, Interventions & a New Framework

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In collaboration with Stress Therapy Solutions, Inc.

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Founder, BrainMaster Technologies Inc.

Disclaimer:

Nothing to disclose

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What to Expect Today

01 Defining pain — biopsychosocial & IASP 2020

02 Introducing Brain Oscillatory Tensegrity

03 Triple network & pain pathways

04 Expert speaker presentations

05 EEG case studies: Sufi, cold pressor & Dessa

06 Neurofeedback protocols & clinical practice

Introducing Today's Speakers

Thomas F. Collura

Ph.D., MSMHC, QEEG-D, BCN | Founder, BrainMaster Technologies

EEG biomarkers for pain, sLORETA imaging. Tom, Erik and Penijean's Sufi EEG case is one of the most dramatic demonstrations of voluntary pain control ever recorded.

Erik Peper

Ph.D. | Professor Emeritus, SFSU

Biofeedback, breathing and holistic health. Erik demonstrates how feeling safe and slow exhalation modulate pain through vagal pathways.

Penijean Gracefire

LMHC, BCN, QEEG-D | Mindfulness & Neurofeedback

Emotional pain, sLORETA Z-score protocols and romantic resiliency. Pioneering work with Dessa showed NFB can release pathological emotional pain loops.

Richard McAlister

DC, DAAPM, BCN, QEEG-DL

Richard is exploring pain modulation and modeling cortical signatures of pain suppression in a Sufi. The hope is to create a z-score database using z-builder for neurofeedback applications in chronic pain.

> *An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.*

— International Association for the Study of Pain (IASP), 2020 Revised Definition

Six Key Notes from IASP 2020

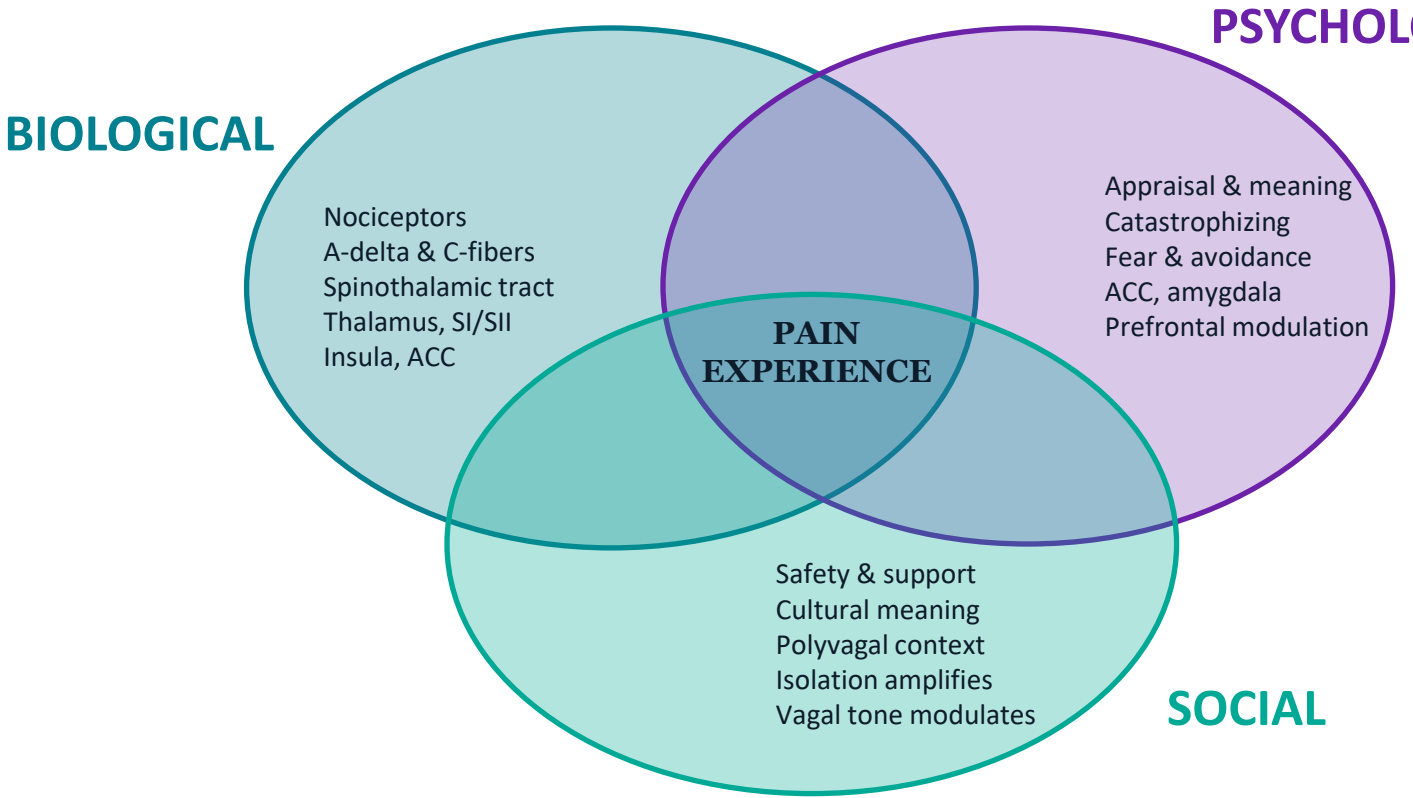
- Pain is always a personal experience influenced by biological, psychological, and social factors
- A person's report of pain should always be respected
- Verbal description is only one way to express pain — inability to communicate does not negate it
- Pain and nociception are different — pain cannot be inferred solely from sensory neuron activity
- Pain usually serves an adaptive role but can adversely affect function and well-being
- People learn the concept of pain through life experiences

Types of Pain & How They Will Be Demonstrated Today

Nociceptive	Neuropathic	Nociplastic	Affective / Emotional
Actual or threatened tissue damage (somatic or visceral). Arthritis, burns, injuries. A-delta & C-fibers.	Lesion or disease of the somatosensory nervous system. Sciatica, diabetic neuropathy. Central sensitisation.	Altered nociception with no clear tissue damage. Fibromyalgia, IBS. The pain system itself is the problem.	The 'second arrow' — distress and suffering added on top of physical sensation. Modulated via non-reactive awareness.
Demonstrated by: Sufi- Self Piercing Bill Brubaker— burning/ freezing sensation from cold.	**Demonstrated by:** NA: Nerve damage cases — altered sensory processing	**Demonstrated by:** Dessa — body symptoms (fatigue, chest tightening) without clear tissue cause	**Demonstrated by:** Strongly reduced in trained Sufi / Dessa states via alpha-dominant brain states

ACUTE (< 3 months): adaptive, protective, recoverable | CHRONIC (> 3 months): maladaptive brain restructuring — BOT architecture collapse

Pain is Always Three Things at Once



Key Insight

Same nociceptive input → dramatically different pain experience depending on context, meaning and safety.

Peper et al., 2025

Is Pain an Emotion?

FIRST ARROW

Physical / Sensory Pain
Nociception → Spinal cord → Brain

+

SECOND ARROW

Emotional / Mental Suffering
Fear, catastrophising, identity fusion

Yes — by definition

IASP 2020 explicitly defines pain as 'sensory AND emotional'. It cannot be one without the other.

ACC & Insula

The anterior cingulate cortex processes pain unpleasantness; the insula integrates bodily feeling with emotional meaning.

We can train the 2nd Arrow

Mindfulness, neurofeedback and breathing directly target emotional amplification — even when nociception is unchanged.

Johnston (1999) — "Why We Feel" | Sallatha Sutta — Two Arrows Model | Nicolardi et al. (2022)

Introducing

Brain Oscillatory Tensegrity

Brubaker & Collura, 2026 — BrainMaster Technologies Perspectives in Neurofeedback

The Core Proposition

The stability of conscious, adaptive cognition — including the ability to process and release pain — is held in the dynamic, distributed balance of oscillatory phase relationships across time. This balance can be understood through the structural principles that Buckminster Fuller called tensegrity.

Pre-stress	Load distribution	Elastic recovery	Failure modes
E/I balance holds the network at the edge of criticality	Pain signals redistributed across three networks	Healthy brains spring back — chronic pain loses this	Collapse = chronic pain, anxiety, identity fusion

Brain Oscillatory Tensegrity

The idea that your brain stays healthy and manages pain through a kind of invisible balancing act — different brain regions constantly pulling and pushing against each other through electrical rhythms, the way the ropes and poles of a tent hold each other in tension to keep the whole structure standing.

When that balance is working properly, pain signals get processed and released. When something disrupts it — like chronic pain, emotional trauma, or prolonged stress — the structure loses its flexibility, and the brain gets stuck, unable to let go of the pain signal.

From Fuller's Structures to Brain Architecture

Buckminster Fuller's Tensegrity

Structural integrity held through COMPRESSION + TENSION

- Pre-stress maintains stability without rigid connections
- Load distributed globally — not locally
- Flexible yet strong — elastic recovery under force
- Remove one cable → global structural could collapse if new stress does not get absorbed
- Deformation under load → springs back when released

In the Brain...

Compression strut	→	Lateral pain pathway (SI/SII) Carries raw nociceptive load
Tension cable	→	Medial pathway (ACC, insula) Distributes suffering globally
Pre-stress mechanism	→	Descending inhibitory circuit PAG → spinal cord modulation
Elastic recovery	→	SN-DMN anticorrelation Healthy networks spring back

The Triple Network — The Brain Oscillatory Tengegrity Architecture

Default Mode Network (DMN)

Key Regions:

mPFC, PCC
Hippocampus
Angular gyrus

In Pain:

Self-narrative, identity, rumination, resting state

Brain Osc. Teng. Role:

Tension element — continuously loaded in chronic pain

Salience Network (SN)

Key Regions:

ACC, Insula
Amygdala
Thalamus

In Pain:

Detects threat & relevance
Switches DMN/CEN
Processes suffering

Brain Osc. Teng. Role:

Dynamic switch — pre-stress regulator between networks

Central Executive Network (CEN)

Key Regions:

DLPFC
Posterior parietal
OFC inhibitory

In Pain:

Cognitive control
Descending pain inhibition
Top-down modulation

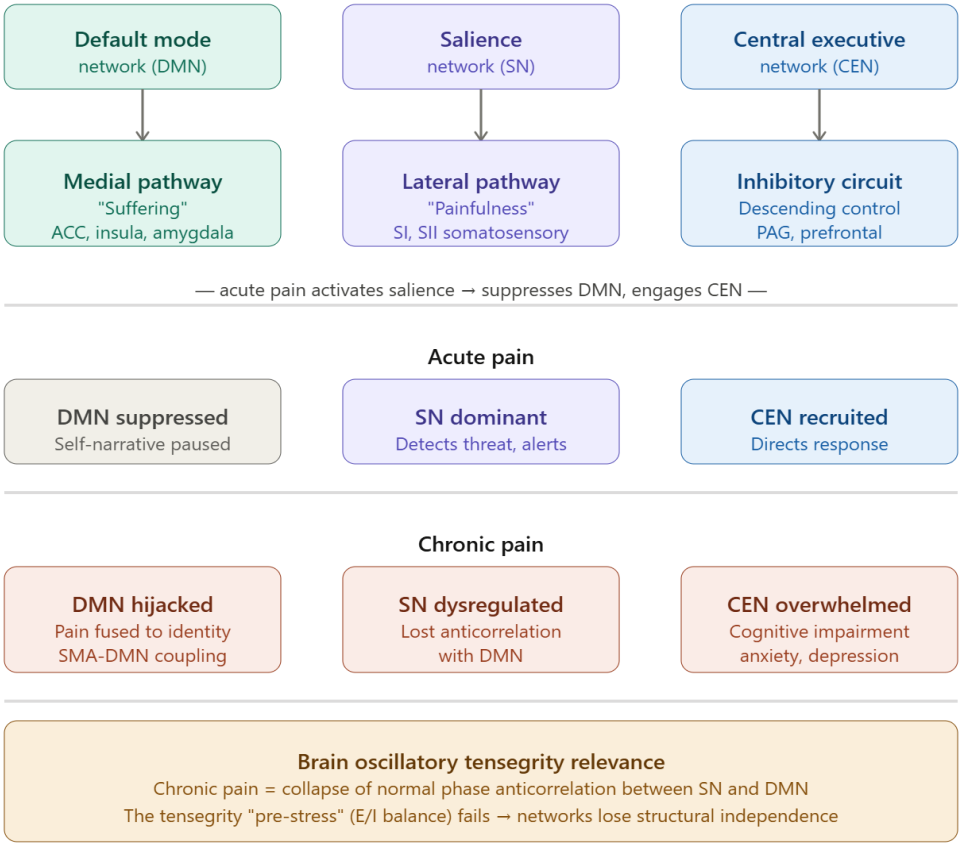
Brain Osc. Teng. Role:

Inhibitory authority — restores pre-stress when functioning

Menon (2011) — Triple Network Model | De Ridder et al. (2022) — Pain & the Triple Network

The Pain Matrix Overlaps the Triple Network

All three cases today — Sufi, cold pressor and Dessa — activated the pain matrix (ACC, thalamus, insula) AND the triple network to varying degrees



The pain matrix is a separate system that overlaps the triple network model | Menon (2011) | De Ridder et al. (2022)

When Tensegrity Holds — and When It Collapses

ACUTE PAIN — Tensegrity Intact

- SN detects nociception → suppresses DMN
- CEN recruited → directs pain response
- Normal SN-DMN anticorrelation maintained
- Alpha gating over sensorimotor cortex intact
- Descending inhibitory pre-stress active
- Structure elastic → springs back on recovery

→ CHRONIFICATION

CHRONIC PAIN — Tensegrity Collapsed

- SN-DMN anticorrelation LOST — co-activated
- Somatosensory cortex phase-locked to DMN
- Brain at rest = brain in pain
- Alpha gating over sensorimotor cortex lost
- CEN loses descending inhibitory authority
- Pain identity fusion — structural deformation permanent

"Chronic pain is not pain that has lasted a long time. It is a brain that has restructured itself around pain." — Brubaker & Collura, 2026

New Terms

Temporal Restructuring*

The process by which BOT training shifts the brain's intrinsic timing relationships — reorganizing phase coherence and oscillatory sequencing across distributed networks — enabling more efficient integration of information and restoration of optimal self-regulation.

Crystals in Time*

The emergence of stable, low-entropy neural oscillatory patterns that, once established through BOT training, persist and self-maintain across time — reflecting the brain's capacity to crystallize and preserve optimized states of self-regulation.

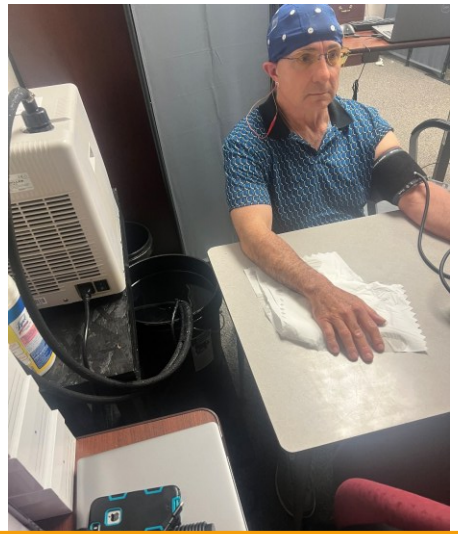
*Definitions subject to change.

Who Felt More Pain?

Three individuals. *Three different pain states.* *Three different brain responses.*

The Sufi

Voluntary piercing
Minimal pain & suffering
Alpha-dominant EEG
BOT: reconfigured



Bill — Cold Pressor

Cold water immersion
Acute nociceptive pain
Alpha suppression EEG
BOT: tensegrity loading

Dessa

Romantic heartbreak
Emotional pain / suffering
DMN-SN coupling
BOT: phase-lock failure

EEG Suppression vs Expression

Sufi EEG: SUPPRESSION

- Strong alpha dominance (high amplitude, hyper-coherent, resembling eyes-closed resting state)
- Maintained or increased alpha throughout the painful event
- Cortical relaxation, sensory disengagement, minimal processing of the stimulus
- Brain Osc. Teng.: Voluntary redistribution of oscillatory load — awareness network de-activated

Cold Pressor EEG: EXPRESSION

- Alpha suppression (desynchronization) + increased beta/gamma during acute tonic pain
- Reduced alpha power, heightened cortical activation in pain matrix areas
- Normal engagement with sensory/emotional processing of ongoing cold pain
- Brain Osc. Teng.: Tensegrity under acute load — architecture stressed, not yet collapsed

RESULT: The Sufi demonstrated trainable alpha-dominant state. Cold pressor showed typical pain-related desynchronization. Alpha enhancement = key NFB target (Collura; Peper).

The Sufi: Voluntary Temporal Restructuring

AAPB Portland 2012 — recorded & presented by Thomas F. Collura, Ph.D.

sLORETA EEG Findings

- **Global gamma DEFICIT**
  Awareness network de-activated
- **Alpha FLOOD**
  Sensorimotor strip + temporal lobes
- **Gamma ABUNDANCE**
  Mesiotemporal — deep inward focus
- **Delta increase**
  Low-frequency coherence globally

BOT Interpretation

The Sufi did not suppress pain. He voluntarily redistributed the oscillatory load into an alternative stable tensegrity configuration — one that excluded pain awareness entirely.

"The structure remained coherent and load-bearing. It simply bore a different load."

Primary pain type: Nociceptive (acute somatic). Affective component: virtually absent. BOT state: alternative stable configuration.

Cold Pressor: Acute Tensegrity Under Load

A direct contrast with the Sufi — same EEG methodology, opposite direction of oscillatory change

What is a Cold Pressor?

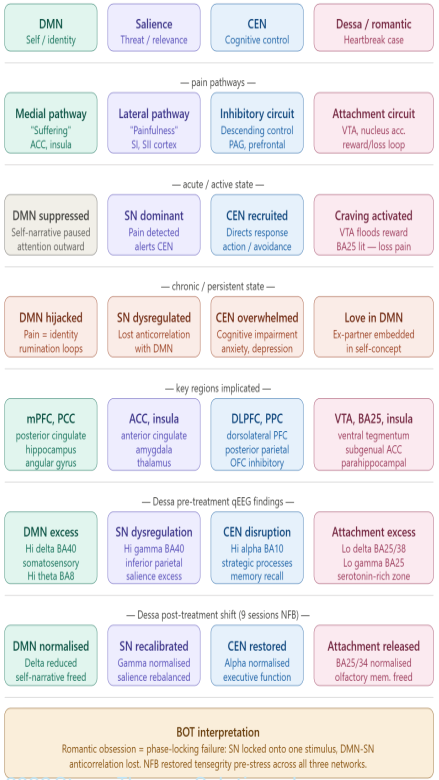
A research tool used to induce pain in a controlled, safe, reproducible way. Safe and reversible. Produces both sensory AND emotional components of pain — ecologically valid.

	Sufi (Suppression)	Cold Pressor (Expression)
Gamma — Awareness Network	DEFICIT — de-activated	ACTIVATION — left temporal
Alpha — Sensorimotor	FLOOD — massive increase	SUPPRESSED — gating lost
DMN-SN Relationship	Voluntary reconfiguration	Beginning to decouple
Right Hemisphere	Globally reorganised	Neutral — not yet recruited
BOT Architecture	Alternative stable state	Tensegrity under stress

BOT: Sufi STRENGTHENED his tensegrity matrix via voluntary reconfiguration. Cold pressor BEGAN COLLAPSING it via nociceptive loading — early stage of what becomes chronic if loading persists.

Dessa: When Pain Doesn't Fit a Category

Gracefire, Dessa & Ohlman — ISNR 2017 | "Can neurofeedback help me fall out of love?"



Dessa's Pain Profile

Her pain does not fit neatly into any single category — yet the body does respond.

- Fatigue
- Chest tightening
- Stomach issues
- Emotional rumination
- Romantic obsession loop

Demonstrated: trainable pain/suffering modulation

Brain Osc. Teng. Interpretation

Romantic obsession = phase-locking failure

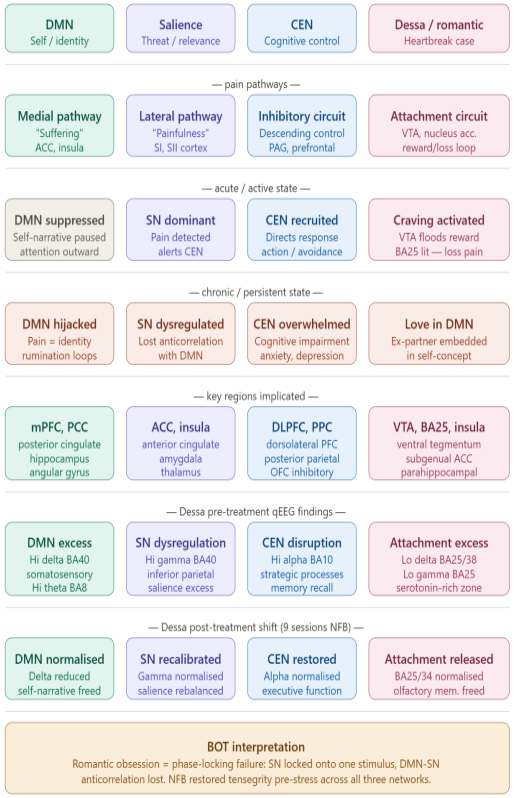
The salience network locked onto her ex-partner as a permanent high-salience stimulus. SN never released → DMN flooded with attachment rumination → SN-DMN anticorrelation lost.

After 9 sessions sLORETA Z-score NFB:

- DMN normalised — self-narrative freed
- SN recalibrated — anticorrelation restored
- CEN executive function restored
- BA25/34 released — olfactory memory freed

Dessa: Mapping Emotional Pain Onto the Triple Network

Pre-treatment qEEG findings, post-treatment normalisation across all three networks after 9 sessions of sLORETA Z-score ***neurofeedback***



DMN
Self / identity
Salience
Threat / relevance
CEN
Cognitive control
Dessa / romantic
Heartbreak case
— pain pathways —
Medial pathway
"Suffering"
ACC, insula
Lateral pathway
"Painfulness"
SI, SII cortex
Inhibitory circuit
Descending control
PAG, prefrontal
Attachment circuit
VTA, nucleus acc.
reward/loss loop
— acute / active state —
DMN suppressed
Self-narrative paused
attention outward
SN dominant
Pain detected
alerts CEN
CEN recruited
Directs response
action / avoidance
Craving activated
VTA floods reward
BA25 lit — loss pain
— chronic / persistent state —
DMN hijacked
Pain = identity
rumination loops
SN dysregulated
Lost anticorrelation
with DMN
CEN overwhelmed
Cognitive impairment
anxiety, depression
Love in DMN
Ex-partner embedded
in self-concept
— key regions implicated —
mPFC, PCC
posterior cingulate
hippocampus
angular gyrus
ACC, insula
anterior cingulate
amygdala
thalamus
DLPFC, PPC
dorsolateral PFC
posterior parietal
OFC inhibitory
VTA, BA25, insula
ventral tegmentum
subgenual ACC
parahippocampal
— Dessa pre-treatment qEEG findings —
DMN excess
Hi delta BA40
somatosensory
Hi theta BA8
SN dysregulation
Hi gamma BA40
inferior parietal
salience excess
CEN disruption
Hi alpha BA10
strategic processes
memory recall
Attachment excess
Lo delta BA25/38
Lo gamma BA25
serotonin-rich zone
— Dessa post-treatment shift (9 sessions NFB) —
DMN normalised
Delta reduced
self-narrative freed
SN recalibrated
Gamma normalised
salience rebalanced
CEN restored
Alpha normalised
executive function
Attachment released
BA25/34 normalised
olfactory mem. freed
BOT interpretation
Romantic obsession = phase-locking failure: SN locked onto one stimulus, DMN-SN
anticorrelation lost. NFB restored tensegrity pre-stress across all three networks.

BOT: Romantic obsession = phase-locking failure. SN locked onto one stimulus, DMN-SN anticorrelation lost. NFB restored tensegrity pre-stress across all three networks.

The Pain Matrix Across All Three Cases

All three cases activated the Pain Matrix (ACC, thalamus, insula) AND the Triple Network to varying degrees. The pain matrix is a separate system that overlaps the triple network.

Sufi	Cold Pressor (Bill)	Dessa
ACC: De-activated (voluntary)	**ACC:** Activated — acute response	**ACC:** Chronically activated (BA25)
Thalamus: Suppressed via alpha gate	**Thalamus:** Engaged — relay active	**Thalamus:** Subcortical loop active
Insula: Disengaged — sensory block	**Insula:** Activated — left temporal	**Insula:** Persistent emotional sensation
Triple Network: All three networks reconfigured into alternative stable state	**Triple Network:** SN dominant, CEN recruited, DMN suppressed — acute load	**Triple Network:** SN-DMN anticorrelation lost, CEN disrupted — phase-lock

De Ridder et al. (2022) | Menon (2011) | Collura (2015) | Gracefire (2017)

Can People Be Trained Not to Feel Pain?

YES

Meditation / Yoga

Lutz et al., 2013; Kakigi et al., 2005

Expert meditators show theta/alpha dominance during pain. Near-complete alpha gating of sensorimotor pain signals — consistent with BOT reconfiguration.

Neurofeedback

Jensen et al., 2013; Caro & Winter, 2001

60-80% reduction in chronic pain with SMR/alpha training. Alpha-theta at Cz/Pz: 70% fibromyalgia relief. sLORETA ACC-insula coherence: emerging.

The Sufi — BOT Model

Collura (2015) AAPB

Voluntary reconfiguration of oscillatory architecture to completely exclude pain awareness. Gamma deficit, alpha flood, mesiotemporal gamma. Years of practice — can NFB compress this?

How to Treat Pain

A multi-modal view from today's expert panel — guided by the BOT framework

Erik Peper
Mind-Body Practices

- Slow exhalation — 6 breaths/min restores vagal tone
- Positive imagery & safety cues reduce amygdala amplification
- Breathing as pre-stress restoration protocol
- Body awareness & posture biofeedback
- HRV training for descending inhibition

Thomas Collura
Alpha Training (NFB)

- Alpha enhancement across sensorimotor strip
- Mirrors the Sufi's voluntary alpha flood
- sLORETA Z-score targeting pain matrix regions
- BrainMaster live sLORETA for real-time imaging
- Alpha-theta protocol at Cz/Pz for fibromyalgia

Penijean Gracefire
sLORETA Training

- Connectivity-based Z-score NFB
- Target ACC, insula, mPFC simultaneously
- Restore DMN-SN anticorrelation
- 9 sessions demonstrated measurable change (Dessa)
- Personalise via qEEG baseline

Emerging Modalities
PEMF & PhotoBioModulation

- Pulsed Electromagnetic Field (PEMF) therapy
- PhotoBioModulation — light-based pain relief
- Both influence oscillatory coherence
- Complementary to NFB approaches
- Audience input welcome!

Peper et al. (2025) | Jensen et al. (2013) | Caro & Winter (2001) | Collura (2015) | Porges (2021)

BOT-Informed Neurofeedback Targets

Target the oscillatory ARCHITECTURE, not just individual frequencies

Restore DMN-SN Anticorrelation

Sites:

ACC, PCC
BA 24, 32, 23, 31
mPFC BA 9, 10

Why:

Most architecturally fundamental — restores the push-pull tension cable that collapses in chronic pain

De Ridder et al. (2022)
sLORETA Z-score NFB

Restore Alpha Sensorimotor Gate

Sites:

C3/C4, Cz
Sensorimotor strip
Temporal lobes

Why:

Alpha over SM cortex = the oscillatory gate. Sufi showed alpha flood = pain suppression. Loss of this gate = nociception propagates globally

Jensen et al. (2013)
Caro & Winter (2001)

Re-establish CEN Inhibitory Authority

Sites:

DLPFC
Posterior parietal
BA 9, 10, 46

Why:

CEN provides top-down descending inhibitory pre-stress. Restoring prefrontal function restores the pre-stress mechanism of the tensegrity

Walker (2011)
Beta suppression 50%

Release Somatosensory -DMN Coupling

Sites:

SI/SII coherence
with mPFC
Connectivity-based

Why:

Pathological phase-locking between SI/SII and DMN = pain identity fusion. Reducing coherence addresses the architectural deformation directly

Baliki et al. (2014)
Emerging connectivity NFB

Personalise via qEEG baseline — protocol selection should match the individual's specific oscillatory failure pattern

What We Have Covered Today

01

Pain is always biopsychosocial

Biological, psychological and social factors are inseparable. IASP 2020 confirmed this as the gold standard definition.

02

Pain is an emotion — the 2nd arrow is trainable

Johnston's two-step model: we cannot always avoid the first arrow but can train to reduce the second via NFB, breathing and reframing.

03

Chronic pain = oscillatory tensegrity collapse

The DMN-SN anticorrelation is lost. Somatosensory cortex fuses to DMN. Pain becomes identity. BOT explains chronic pain resistance to treatment.

04

The Sufi showed voluntary reconfiguration is possible

Real EEG data: gamma deficit across awareness network, alpha flood over sensorimotor strip — voluntary BOT reconfiguration. NFB may compress this.

05

Target the architecture, not just the frequency

BOT-informed NFB targets oscillatory structure: restore DMN-SN anticorrelation, restore alpha gating, re-establish CEN inhibitory authority.

06

Safety restores the pre-stress

Feeling safe is not just psychological — it is the physiological substrate of the descending inhibitory pre-stress mechanism. Breathing works.

Key Sources

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Full APA reference list available: apa_references_brubaker_collura.md

Thank You

Questions, Discussion & Panel Q&A

"The goal of neurofeedback in chronic pain is not to silence the pain signal. It is to restore the load-bearing architecture of the brain so that the pain signal can once again be processed, released, and forgotten."

— Brubaker & Collura, 2026

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Brain Oscillatory Tensegrity — Brubaker & Collura (2026)